results show that sham smoking of an unlit cigarette or smoking without inhaling has no effect but that inhalation of cigarette or pipe smoke increases the cardiac output by 40% to 100% above the control values. Blood-pressure and heart rate both rose slightly when the output increased. A. Kershbaum and colleagues[3] showed that the output of noradrenaline-like substances increased 17% with cigarette smoking except in adrenalectomized subjects, and so it seems probable that the increase in cardiac output is secondary to nicotine stimulation of the adrenal medulla via the sympathetic ganglia.

It might be tempting to regard this increase of output with smoking as a good thing, but in fact there are sound reasons for believing that tobacco does only harm to the cardiovascular system. Intermittent claudication, for example, is virtually unknown in non-smokers,[4] and coronary-artery disease is commoner in heavy cigarette smokers than in others. Nicotine reduces the coronary blood flow in atherosclerotic rabbits,[5] and smoking in men with coronary artery disease gives abnormalities in the electrocardiogram[6] and the ballistocardiogram.[7] What we should now try to determine is whether smoking can by itself cause atherosclerosis and if so by what mechanism.

HARDNESS OF WATER AND CARDIOVASCULAR DISEASE

In Japan,[1] the U.S.A.,[2 3] and Great Britain[4 5] local differences in mortality from cardiovascular disease are negatively associated with the hardness of drinking-water: that is to say, the softer the water the higher the death rate. In England and Wales about one-fifth of the variation in cardiovascular mortality between the county boroughs appears to be "explained" by this difference in the hardness of the water: this is equivalent to about 7,000 deaths per annum in men aged 45 to 64 alone. Not surprisingly regional differences in cardiovascular morbidity also are associated with hardness of water supplies. Such differences have been found in Japan for cerebrovascular disease, and now a recent report[6] on a hospital in-patient inquiry shows trends in admissions for "arteriosclerotic heart disease" which follow to some extent the geographical distribution of hard and soft waters. In the soft-water areas of Lancashire and Yorkshire admission rates for this disease group are almost double those in the hard-water area of the Midlands. Hospital admission rates, however, are not a reliable index of actual morbidity in a locality, and they must be interpreted with caution. Studies of clinical prevalence and post-mortem surveys specifically directed to this problem have now been started in Britain and in Sweden.

The "water factor," as it may be called, in cardiovascular disease then apparently is operating in several countries. Moreover, in Britain at least it seems to be independent of socio-economic and other environmental differences between the big towns.[4 5] Whatever it is, its effects are sizable: even a small contribution to cardiovascular mortality has important public-health implications. The two main questions are as yet unanswered. The principal groupings within cardiovascular mortality —that is, cerebrovascular disease, coronary heart disease, and "myocardial degeneration"—are all highly significantly correlated with water hardness. It is not yet known whether the association is with one of the pathological components of degenerative cardiovascular disease—atheroma, arterial occlusion, hypertension—or with some non-specific factor in cardiac failure.

The other question relates to the chemical nature of the water factor, and this is proving very difficult to identify. Analyses for trace elements by emission spectroscopy have so far shown no important differences between hard and soft English drinking-waters. Further studies are now in progress both here and in the U.S.A. From the data already available, there is no evidence that mineral contaminants from water pipes (e.g., lead, zinc, iron, etc.) are involved. It is thus still impossible to say whether hard water is in some way protective, or if some characteristic of soft water is harmful.

VIRUSES AT THE LISTER

When Sir Howard Florey opened a new virus laboratory at the Lister Institute's country branch at Elstree on May 22 he marked in a physical way a change of emphasis already apparent there. Until quite recently the sole tasks of the Institute's virus vaccines department under Dr. Colin Kaplan were the production, on behalf of the Ministry of Health, of smallpox vaccine for use in England, Wales, and Northern Ireland, and research on vaccinia virus. This limitation of activity was dictated by the Therapeutic Substances Regulations which, for safety reasons, forbid the handling of another virus in a laboratory where smallpox vaccine is being made. This caution is understandable, but it has tended to restrict the department's research programme. With the present rapid extension of knowledge of viruses, it is clearly vital that experienced units such as the one at the Lister should have every chance to range widely in their investigations, and this the provision of the new laboratory will allow. From being the smallpox vaccine department, the Unit was rechristened this year the department of virus vaccines —an earnest of what was to come—and now the necessary physical conditions have been provided.

The new laboratory consists of two completely self-contained suites, with separate ventilation. One suite will be for the production of virus vaccines. It can be used to produce only one type of vaccine at a time, but different vaccines can be made in it in rotation. The second suite is for research on viruses, and because the two suites are bacteriologically separate it will now

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